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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.
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EXAMINER	
GOLDBERG, J	

ART UNIT	PAPER NUMBER
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Please find below and/or attached an Office communication concerning this application or proceeding.

Commissioner of Patents and Trademarks

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1- File Copy

Office Action Summary	Application No.	Applicant(s)	
	09/613,887	HOGAN, KIRK	
	Examiner	Art Unit	
	Jeanine A Goldberg	1655	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *09 August 2001* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *21-41* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *21-41* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____ .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: .

DETAILED ACTION

1. This action is in response to the papers filed August 9, 2001. Currently, claims 21-41 are pending. All arguments have been thoroughly reviewed but are deemed non-persuasive for the reasons which follow.

2. Any objections and rejections not reiterated below are hereby withdrawn.

3. This action contains new grounds of rejection necessitated by amendment.

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

4. Claims 21-41 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

A2) Claims 21-41 are indefinite over the recitation "known". It is unclear what is meant by known. The term "known" in claim 21, 32, 37 is a relative term which renders the claim indefinite. The term "known" is not defined by the claim, the specification does not provide a standard for ascertaining the requisite degree, and one of ordinary skill in the art would not be reasonably apprised of the scope of the invention. Known may be relative to the time of filing the application, time of publishing the patent, time the reader is reading the publication. Thus, known is not clear and definite.

B2) Claims 21-31 are indefinite over the recitation "before completion of said surgery" because it is unclear what constitutes completion of surgery. Completion may be considered when the doctor is no longer performing work, once the patient has completed rehabilitation or when the patient has left the hospital premise.

C2) Claims 21-31 are indefinite because it is unclear whether perioperative subject has been defined as "a patient who is scheduled for surgery but before completion of said surgery" or whether the claim merely requires the assay to be performed during this time period. Based upon the interview conducted, the examiner was interpreting the claims to require taking a sample from a perioperative patient, wherein a perioperative patient is a patient scheduled for surgery but before completion of said surgery. The claim however, does not appear to require this limitation but instead requires that the assay be performed during this time period.

D2) Claims 21-31are indefinite because the claims do not recite a positive process step which clearly relates back to the preamble. The preamble states that the method is for screening a patient perioperatively to determine a risk for surgical complications but the final process step is subjecting the sample to an assay for detecting two or more genetic markers to generate a genomic profile for use in selecting a perioperative course of action. Therefore the claims are unclear as to whether the method is a method of determining risk for surgical complications or selecting a perioperative course of action. "For use in selecting a perioperative course of action" is not an active method step. In order to meet the limitation of the claims, it is unclear whether an assay for two or more genetic markers is required or whether these markers

must be indicative of surgical risk. Claim 31 appears to require such limitations, indicating that surgical risk is not required in Claim 21.

E2) Claims 32-36 are indefinite because the claims do not recite a positive process step which clearly relates back to the preamble. The preamble states that the method is for selecting conditions for a surgical procedure by screening a patient perioperatively to determine risk for a surgical complications associated with known genetic variations but the final process step is subjecting said subject to a surgical procedure. Therefore the claims are unclear as to whether the method is a method for selecting conditions for a surgical procedure by screening a patient perioperatively to determine risk for a surgical complications associated with known genetic variations or a method of subjecting a subject to a surgical procedure. "For use in selecting a perioperative course of action" is not an active method step. Thus, the final process step and the preamble do not correspond.

F2) Claims 37-41 are indefinite because the claims do not recite a positive process step which clearly relates back to the preamble. The preamble states that the method is for screening a patient perioperatively to determine risk for a surgical complications from known genetic variations but the final process step is subjecting said subject to an assay for detectin two or more genetic markers. Therefore the claims are unclear as to whether the method is a method for screening a patient perioperatively to determine risk for a surgical complications associated with known genetic variations or a method of assaying two or more genetic markers. "For use by a physician in selecting a perioperative course of action" is not an active step. It is unclear what limitations are

added to the claim by this "for use" statement. Claim 41 requires "using said profile for selection of conditions for a surgical procedure carried out on said patient". This is not an active step which clearly sets forth how to "use" this information. Thus, the final process step and the preamble do not correspond.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

5. Claims 21, 24, are rejected under 35 U.S.C. 102(b) as being anticipated by Connors et al (Biochimica et Biophysica Acta, Vol 1407, page 185-192, 1998).

This rejection is applicable because the final step of the claim does not require determining surgical risk.

Connors et al (herein referred to as Connors) teaches a method of testing sera from patients with transthyretin type amloidosis (ATTR). The samples were tested prior to and following surgery. Fifteen variants of plasma protein transthyretin were examined and found in 74 of the 110 serum samples tested. Connors states that TTR analysis by IEF was performed on serum samples obtained at multiple time points before and after surgery from each of more than 10 patients who underwent liver transplantation as treatment for their disease. The appearance of a TTR variant was apparent pretransplantation and absent at all time points tested postsurgery.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

6. Claims 21-28, 31-35 are rejected under 35 U.S.C. 103(a) as being unpatentable over Quane et al (Human Molecular Genetics, Vol 3, No. 3, page 471-476, 1994).

Quane et al (herein referred to as Quane) teaches the detection of novel common mutations in ryanodine receptor gene (RYR1) in malignant hyperthermia (MH). Malignant hyperthermia (MH) is triggered in susceptible people by all commonly used inhalation anesthetics. Quane has identified Gly341Arg mutation which accounts for approximately 10% of Caucasian MHS cases (abstract). Quane specifically teaches that once an individual is diagnosed as being susceptible to MH, the anesthetics which trigger this syndrome can be avoided. Quane also teaches that Arg615Cys is a substitution found in 3-5% of human MH families investigated (page 472, col. 1); Arg163Cys is a substitution found in 2-3% of MHS cases. Furthermore, three other rare mutations have been reported in the RYR1 gene which are in three isolated MHS and/or CCD cases. Quane teaches that patients which have not been indicated as MH normal should always be considered MHS clinically to avoid any possibility of the individual reacting to a triggering agent during anesthesia. Misdiagnosis of MHS individual as

MHN can be lethal if such a patient is exposed to triggering agents (page 474, col. 1). Quane teaches that the mutation reported satisfies the genetic criteria necessary for demonstration of a causal mutation and as such this mutation should be of significant value for MHS diagnosis by genetic means (page 474, col. 1).

While Quane does not explicitly state, that samples were taken from preoperative patients, Quane specifically suggests that "once an individual is diagnosed as being susceptible to MH, the anesthetics which trigger this syndrome can be avoided (page 471, col. 2).

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have sampled patients prior to subjecting the patient to anesthetics to determine whether they were at risk of MH. The ordinary artisan would have clearly recognized the benefit of testing an individual prior to surgery and subjection to the anesthesia for known genetic markers associated with a condition which was triggered by anesthetics. The ordinary artisan would have been motivated to test these individuals prior to surgery for the expected benefit of determining whether the patient possessed any mutations which were linked to the known condition of MH to avoid any fatal reaction to the anesthesia.

7. Claims 21, 26-28, 31-34, are rejected under 35 U.S.C. 103(a) as being unpatentable over Acta Anaesthesiologica Scandinavica (Vol 39, page 139-141, 1995) and La Du (Cellular and Molecular Neurobiology, Vol 11, No. 1, page 79-89, 1991).

Acta Anaesthesiologica Scandinavin referred to as AAS) teaches that certain variants have a dramatic degree of resistance to the drug, succinylcholine (SC), because they destroy it so rapidly. AAS teaches that individuals show no regular metabolic disoder unless SC or mivacurium is given such that the condition is provoked. BchE mutations are dibucaine resistant, fluoride resistant or silent.

La Du et al (herein referred to as La Du) teaches butyrylcholinesterase variants which have been found in individuals who have responded abnormally to the muscle relaxant succinylcholine. Variants with increased activity are resistant to succinylcholine and may require two or three doses to achieve the desired state of paralysis (page 80).

Neither AAS nor La Du explicitly teaches, that samples were taken from preoperative patients, however, AAS and La Du teach of the benefits for individual treatment.

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have sampled patients prior to subjecting the patient to succinylcholine, for example, to determine whether they were resistant to the drug. The ordinary artisan would have clearly recognized the benefit of testing an individual prior to surgery and subjection to succinylcholine for known genetic markers associated with a condition which causes certain individuals to have a dramatic degree of resistance to the drug because they destroy it so rapidly requiring two or three doses to achieve the desired state of paralysis. The ordinary artisan would have been motivated to test these individuals prior to surgery for the expected benefit of

determining whether the patient possessed any mutations which were linked to the known condition of rapid metabolizers to avoid ineffective treatment.

8. Claims 21, 26-28, 31-34, are rejected under 35 U.S.C. 103(a) as being unpatentable over Pharmacogenetics (Chapter 4, pages 309-326, IDS #201) and Evans et al (Science, Vol. 286, pages 487-491, October 1999).

Pharmacogenetics teaches polymorphisms of desbrisoquine hydroxylase (Cytochrome P4502D6). The structures of CYP2D gene clusters are provided. The poor metabolizers are depicted. Pharmacogenetics teaches that for drugs such as codeine and encainide it is the PM subjects who may experience therapeutic failure (page 317, col. 1). Codeine is ineffective analgesic in the 5-10% of the population who have a PM phenotype. The discovery and identification of each of these conditions has saved some lives and may prevent future fatalities or morbidities.

Evans et al (herein referred to as Evans) teaches the drug-metabolizing enzyme debrisoquine hydroxylase (CYP2D6) is polymorphic. Evans teaches that "inherited differences in drug-metabolizing capacity are generally monogenic traits and their influence on the pharmacokinetices and pharmacologic effects of medications is determined by their importance for the activation or inactiviation of drug substrates (page 487, col. 2). Evens also teaches "the effects can be profound toxicity for medications that have a narrow therapeutic index and are inactivated by a polymoprhic enzyme (for example, mercaptopurine, azathioprine, thioguanine, and fluorouracil) or reduced efficacy of medications that require activation by an enzyme exhibiting genetic

polymorphism (such as codine)" (page 487, col. 3). Evans illustrates in Figure 2, drug-metabolizing enzymes known to exhibit genetic polymorphisms with incontrovertible clinical consequences. Further, severe and potentially fatal hematopoietic toxicity that occurs when thiopurine methyltransferase-deficient patients are treated with standard doses of azathioprine or mercaptopurine. Evans teaches that "many opioid analgesics are activated by CYP2D6 rendering the 2-10% of the population who are homozygous for nonfunctional CYP2D6 mutant alleles relatively resistant to opioid analgesic effects. Thus is it not surprising that there is remarkable interindividual variability in the adequacy of pain relief when uniform doses of codeine are widely prescribed" (page 489, col. 1). Evans teaches that individualizing drug dosages can improve clinical outcome (page 491, col. 1).

Neither Pharmacogenetics nor Evans explicitly teaches, that samples were taken from preoperative patients, Pharmacogenetics and Evans teaches of the benefits for individual treatment.

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have sampled patients prior to subjecting the patient to anesthetics to determine whether they were at risk of being a poor metabolizer of a drug, namely codeine. The ordinary artisan would have clearly recognized the benefit of testing an individual prior to surgery and subjection to codeine for known genetic markers associated with a condition which causes certain individuals to be poor metabolizers. The ordinary artisan would have been motivated to test these individuals prior to surgery for the expected benefit of determining whether the patient

possessed any mutations which were linked to the known condition of poor metabolizers to avoid ineffective analgesia or therapeutic failure.

9. Claims 21, 28, 30-32, 36 are rejected under 35 U.S.C. 103(a) as being unpatentable over Poort et al (Blood, Vol 88, No. 10, page 3698-3703, 1996).

Poort et al (herein referred to as Poort) teaches an 20210 AG gentoype of the prothrombin gene which is a candidate for venous thrombosis in patients. It is well known in the art that venous thromboembolism can occur without apparent cause, after surgical procedures or trauma. Poort also teaches that factor V Leiden is the most common hereditary risk factor for thrombosis.

Poort does not explicitly teach, that samples were taken from preoperative patients. However, Poort teaches two genetic markers which are associated with thrombosis.

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have sampled patients prior to subjecting the patient surgery to determine whether they were at risk of thrombosis. The ordinary artisan would have clearly recognized the benefit of testing an individual prior to surgery for known genetic markers associated with thrombosis which is often triggered by surgery or thrombosis. The ordinary artisan would have been motivated to test individuals prior to surgery for the expected benefit of determining whether the patient possessed any mutations which were linked to the known condition to avoid and be

aware of potential side effects which should be part of the consideration of the patient when deciding to undergo surgery.

10. Claims 29-30, 36-41 are rejected under 35 U.S.C. 103(a) as being unpatentable over Quane et al (Human Molecular Genetics, Vol 3, No. 3, page 471-476, 1994) as applied to Claims 21-28, 31-35 above; Acta Anaesthesiologica Scandinavica (Vol 39, page 139-141, 1995) and La Du (Cellular and Molecular Neurobiology, Vol 11, No. 1, page 79-89, 1991) as applied to Claims 21,26-28, 31-34 above; Pharmacogenetics (Chapter 4, pages 309-326, IDS #201) and Evans et al (Science, Vol. 286, pages 487-491, October 1999) as applied to Claims 21, 26-28, 31-34 above; and Poort et al (Blood, Vol 88, No. 10, page 3698-3703, 1996) as applied to Claims 21, 28, 30-32, 36 above.

None of the cited references specifically discuss testing multiple known markers which are associated with different conditions, i.e. known genetic markers into a single assay for determining whether individuals are at risk during surgical procedures.

However, the state of the art with relation to known polymorphisms and detecting the polymorphisms as indicative of certain disease which either trigger episodes when exposed to anaethetics, or are poor metabolizers or potentially cause thrombosis are well known.

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have modified the vast number of teachings, as exemplified by the extremely voluminous Information Disclosure Statement filed, to

screen individuals prior to surgery for several genetic markers which are indicative of any number of conditions which are caused by anaethesia or are a result of anaethesia. The ordinary artisan would have recognized that the art provides a large number of single nucleotide polymorphisms or other variations which are indicative of conditions. The benefit of screening individuals for several of these prevalent mutations which are related to surgery would have allowed the anaetheseologist to determine whether plausible substitutes may be provided to patients which would not cause these conditions to arise. Specifically, detection of RYR1 polymorphisms which are associated with MH would indicate to the anaetheologist that drugs which trigger the episodes should be avoided. Moreover, codeine should be administered with care to individuals having certain BchE mutations. Combining more than one screening method to determine the genomic profile of a patient would have provided the anaetheseologist with a more complete picture of the patients genetic make-up. As suggested in many of the articles, individual treatment and screening is ideal for analysis of the genetic make-up of patients.

Conclusion

11. No claims allowable.

12. The prior art made of record and not relied upon is considered pertinent to applicant's disclosure.

A) Brandt et al (Human Molecular Genetics, Vol. 8, No. 11, pages 2055-2062, 1999) teaches that 21 RYR1 mutations have been identified which account for more

than 50 of the families with susceptibility to MH. Brandt teaches that the genetic testing may be used to determine whether individuals are likely to have MH.

B) Ciccone et al (herein referred to as Ciccone) teaches that "In anaesthesia, our preoperative assessment includes prescribed medications and allergies to drugs. We also consider factors, either directly or indirectly, which may influence responses to drugs, such as age, genetic history, metabolic phenotype,..." (page 255-256).

C) Monnier et al (herein referred to as Monnier) teaches a novel mutation in CACLN1A3 which segregate perfectly with the MHS phenotype in a French family. The substitution of an Arg-His at residue 1086 results in the transition of A for G3333.

D) Jensen et al (Acta Anaesthesiologica Scandnavica, Vol 39, page 150-156) teaches that patients with abnormal BchE often have prolonged apnoea following succinylcholine. Jensen teaches that on should not try to treat the block, but rather keep the patient anaesthetized and ventilated till the usually clinical criteria for full recovery are present. Further, when a clinician is faced with a patient with an apparent abnormal response to succinylcholine, the use of a nerve stimulator is urged.

E) Masterson et al (Br. J. of Anaesthesia, Vol 77, No. 5, page 569-571, 1996) teaches that patients which are likely to mount excessive cytokine responses after surgery may be tested. "Such tests may help anaesthetists to predict outcome or the need for postoperative intensive care. They may also allow us to select the most appropriate anaesthetic, in terms of its ability to modulate cytokine activity, for each patient.

F) Caplan teaches numerous costs of adverse outcomes for anesthesia-related deaths. Among these costs is not only the economic costs, but also non-economic costs.

G) Larson et al (herein referred to as Larson) teaches the preoperative testing for a T to C transition in the codon for amino acid 85 of the beta globin gene. The individual was tested for an unstable Hb variant resulting in congenital hemolytic anemia which has an increased affinity for oxygen. Larson teaches that chronic hemolysis may result in cholelithiasis requiring cholecysterctomy. Perioperative management of this congenital hemoglobinopathy by partial-echange erythrocytapheresis to prevent introperative tissue hypoxia during general anesthesia and cholecystectomy. Lason describes the "perioperative management of a patient, with the unstable, high-oxygen-affinity Hb, HbBryn Mawr, who was deemed at risk for significant tissue hypoxia during general anesthesia and surgery".

H) Hecht et al (Anesth. Analg, Vol. 84, pg. 461-464, 1997) teaches a G1583A mutation in CACNL1A3 which is associated with HypoPP. Hecht also teaches that HypoPP has been identified as a disorder that can predispose a patient to the syndrome of MH which the risk of triggering skeletal muscle contraction and rhabdomyolysis, together with earlier reports of flaccid paralysis aggravated by surgery and general anesthesia, appear to favor regional anesthesia in this population whenever feasible (abstract). Hecht also teaches that MH susceptibility associated with HypoPP and of hypokalemia elicited by regional anesthesia suggests that hybrid anesthetic techniques be avoided (pg. 462, col. 2).

I) Korte et al (Clin. Chem. Lab. Med, Vol. 36, No. 4, pg. 235-240, 1998) teaches to establish a possible "perioperative reference range" for thrombin generation prothrombin fragment F1+2 and fibrin degradation markers were measured (abstract). Korte also teaches that preoperative determination of molecular markers would be helpful in identifying a group of patients at high risk for intraoperative disorder of hemostasis by exclusion of low risk patients (abstract). As seen in Table 2 and Table 3, the results of the detection assay for the two genetic markers were observed (pg. 237).

J) Brandt et al (Hum. Mol. Genetics, Vol 8, No. 11, pg 2055-2062, 1999) teaches screening of approximately 105 MH families for mutations. Despite the extensive number of known mutations in RYR1, "interpretation must be performed with care because lack of the particular mutation segregating in the family does not exclude absence of further independent unknown mutations. Additionally, genetic screening is not yet suitable for routine diagnostics due to the low incidence of each mutation and the vastness of the gene" (pg 2058, col 2).

K) De Stefano et al (New England J. Med, Vol 341, pg 801-806, 1999) teaches screening for two point mutations, one in F 5 Leiden and one in the prothrombin gene which are the most common causes of inherited thrombophilia. Thus, carriers of both of these mutations have an increased risk of recurrent deep venous thrombosis after a first episode and are candidates for lifelong anticoagulation.

13. Any inquiry concerning this communication or earlier communications from the examiner should be directed to examiner Jeanine Enewold Goldberg whose telephone

number is (703) 306-5817. The examiner can normally be reached Monday-Thursday from 7:00AM to 4:30 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Jones, can be reached on (703) 308-1152. The fax number for this Group is (703) 305- 3014.

Any inquiry of a general nature should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Jeanine Enewold Goldberg
October 22, 2001

W. Gary Jones
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